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Understanding the Signs, Symptoms, and Latest Therapeutic Recommendations for NK

MARK A. PAVILACK, MD

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Understanding the Signs, Symptoms, and Latest Therapeutic Recommendations for NK

Faculty

Mark A. Pavilack, MD
Cornea, Cataract, and Refractive Surgeon
Tidewater Eye Centers, an ECP Partner
Portsmouth, VA
Assistant Clinical Professor
Eastern Virginia Medical School
Norfolk, VA

Content Source

This continuing education (CE/CME) activity captures content from a synchronous in-person regional meeting.

Activity Description

This supplement summarizes a discussion on employing classification/staging systems for neurotrophic keratitis in facilitating early diagnosis and intervention.

Target Audience

This certified CE/CME activity is designed for optometrists and comprehensive ophthalmologists.

Learning Objectives

Upon completion of this activity, the participant should be able to:

- **Describe** the etiologies, comorbid diseases, surgical issues, and patient-specific factors that may heighten suspicion for neurotrophic keratitis (NK)
- **Analyze** the relationship between different corneal components that underscores the pathophysiology of NK
- **Perform** diagnostic evaluations that distinguish between dry eye and NK
- **Employ** classification/staging systems for NK in facilitating early diagnosis and intervention
- **Identify** the clinical goals of managing NK by assessing the holistic impact on patients, including disease burden and patient experience

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PRETEST QUESTIONS

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1. Please rate your confidence in your ability to employ classification/staging systems for neurotrophic keratitis (NK) in facilitating early diagnosis and intervention (based on a scale of 1 to 5, with 1 being not at all confident and 5 being extremely confident).

a. 1
b. 2
c. 3
d. 4
e. 5

2. Which of the following factors is NOT an etiology of NK?

a. Long-term contact lens wear
b. Short-term use of topical eye drops
c. Increasing age
d. Diabetes

3. A 64-year-old patient presents with blurred vision in his right eye for 2 months. He has a history of hypertension, congestive heart failure, and facial trauma. Punctate epitheliopathy without stromal haze is observed during the slit-lamp examination. Which patient-specific factors increase the suspicion for NK?

a. Facial trauma and epitheliopathy
b. Hypertension and facial trauma
c. Hypertension, facial trauma, and epitheliopathy
d. Congestive heart failure and facial trauma

4. NK is characterized by the breakdown of the corneal _____ and impaired corneal innervation of the _____ nerve.

a. Epithelium, facial
b. Epithelium, trigeminal
c. Stroma, facial
d. Stroma, trigeminal

5. Which of the following diagnostic tests is used to distinguish between dry eye disease and NK?

a. Corneal sensitivity
b. Corneal staining
c. Schirmer test
d. Tear film osmolarity

6. A 58-year-old patient presents with symptoms of dryness. She has a history of poorly controlled diabetes and has used artificial tears for years. She is a nonresponder to lifitegrast and cyclosporine ophthalmic solutions. Which of the following diagnostic strategies is most appropriate?

a. Perform a workup for dry eye that includes corneal staining and Schirmer testing without use of topical anesthetic
b. Perform a workup for dry eye that includes corneal staining and Schirmer testing with use of topical anesthetic
c. Perform a workup for dry eye that includes corneal staining and sensitivity testing without use of topical anesthetic
d. Perform a workup for dry eye that includes corneal staining and sensitivity testing with use of topical anesthetic

7. NK observed as corneal perforation is graded as stage _____ and stage_____ according to the classification of Mackie and the Neurotrophic Keratitis Study Group (NKSG), respectively.

a. 1, 3
b. 2, 3
c. 2, 4
d. 3, 6

8. A 72-year-old patient presents with blurred vision. During the slit-lamp examination, a central epithelial defect with stromal scarring and corneal hypoesthesia are observed in his left eye. According to the NKSG classification, what is the stage of NK?

a. 3
b. 4
c. 5
d. 6

9. A 54-year-old patient presents with blurred vision and redness. During the slit-lamp examination, punctate epitheliopathy with stromal haze and corneal hypoesthesia (Mackie Stage 2) are observed in her left eye, and punctate epitheliopathy without stromal haze and corneal hypoesthesia (Mackie Stage 1) are observed in her right eye. Telangiectasia and anterior blepharitis are observed in both eyes. According to the NKSG, which of the following treatment recommendations is most appropriate?

a. Recommend a pressure patch and topical antibiotic for left eye
b. Recommend a silicone hydrogel bandage contact lens and topical antibiotic for left eye
c. Recommend a silicone hydrogel bandage contact lens, topical antibiotic, and topical corticosteroid for left eye
d. Recommend a silicone hydrogel bandage contact lens for left eye and a topical antibiotic and topical corticosteroid for both eyes

10. A 2020 study by Murray et al examined the patient experience of NK using a questionnaire and found that patients with NK reported the ocular symptom of _____ only when queried.

a. Eye fatigue
b. Sensitivity to light
c. General discomfort
d. Blurred vision

11. A 78-year-old patient presents with symptoms of dry eye, photophobia, and a loss of interest in driving. In the past, he has been nonresponsive to topical prescription medications for dry eye and preservative-free artificial tears. During the slit-lamp examination, meibomian gland dysfunction, punctate epitheliopathy without stromal haze, and corneal hypoesthesia (Mackie Stage 1) are observed in both eyes. Which of the following management approaches is most appropriate?

a. Recommend thermal pulsation light therapy, blepharoexfoliation, and follow-up in 6 weeks
b. Recommend thermal pulsation light therapy, blepharoexfoliation, an artificial tear ointment at night, and follow-up in 6 weeks
c. Recommend thermal pulsation light therapy, blepharoexfoliation, punctal plugs, and follow-up in 6 weeks
d. Recommend thermal pulsation light therapy, blepharoexfoliation, topical cenegermin ophthalmic solution, and follow-up in 6 weeks

Understanding the Signs, Symptoms, and Latest Therapeutic Recommendations for NK

We have long identified neurotrophic keratitis (NK) in our patients by the loss of corneal sensitivity. Last year, a group of high-level experts in NK formed the Neurotrophic Keratopathy Study Group (NKSG) to look more broadly at the factors underlying NK and examine how we can treat the condition earlier and potentially prevent progression. As a starting point, the group arrived at a definition of NK: "The dysfunction of corneal innervation that results in dysregulation of corneal and/or cellular function. It is characterized by loss of corneal sensation and neuronal homeostasis, leading to eventual corneal epithelial breakdown and ultimately keratolysis if untreated."[1]

The prevalence of NK is difficult to determine, but a recent review of the American Academy of Ophthalmology Intelligent Research in Sight (IRIS) Registry database showed 21.34 cases per 100,000 patients in the United States,[2] giving it rare/orphan disease status.[3] However, it has been shown that because NK is considered rare and its epidemiology is not fully understood, doctors can overlook a diagnosis of NK.[4] In fact, the NKSG found that NK is more prevalent than previously thought.[1]

To ensure we identify and treat NK and prevent damage to the ocular surface, we need to understand how the disease presents. The NKSG's updated definition and staging system provides us a framework to diagnose and treat NK before permanent vision loss occurs, with treatment options for distinct stages of the disease. Here, we will take a comprehensive look at the NKSG's work, as well as NK's pathophysiology, etiologies, comorbidities, diagnostic clues and evaluations, classification and staging, clinical goals for management, and current therapies.

PATHOPHYSIOLOGY AND OVERLAP WITH OSD

We often describe NK simply as a lack of sensation, but that's just one of the manifestations of this condition. The NKSG pointed out that NK makes the cornea more vulnerable to damage by diminishing the response to stimulation.[1] Normally, the nerves help maintain the ocular surface by initiating protective reflexes, such as delivering critical trophic factors to corneal cells for healing. In eyes with NK, dysfunctional corneal sensation sets off a cascade of events, including loss or imbalance of trophic factors needed for epithelial cell proliferation, migration, and differentiation, decreased mitosis of epithelial cells, and impaired corneal healing.[1]

NK also causes secondary alterations of the lacrimal functional unit (LFU). This integrated system—the lacrimal glands, meibomian glands, goblet cells, ocular surface, eyelids, and connecting sensory and motor nerves—maintains homeostasis on the ocular surface. In patients with NK, LFU alterations may differ depending on the disease stage, from an irregular or hazy corneal epithelium to perforation.[1] The tear film and blink rate decrease, which may lead to aqueous deficiency and evaporative dry eye disease (DED), inflammation, and damage to the ocular surface. Doctors notice obvious defects in the epithelium or tear film, but they may not appreciate how restoring corneal innervation or supplying trophic factors could improve these signs and symptoms.

"The Neurotrophic Keratopathy Study Group's updated definition and staging system give us a framework to diagnose and treat NK before permanent vision loss occurs, with treatment options for distinct stages of the disease."

—Mark A. Pavilack, MD

According to the NKSG, this overlap between NK and ocular surface disease (OSD) is one reason that NK's prevalence is underestimated.[1] They noted evidence of some decreased sensitivity in DED and higher rates of NK in patients with OSD who don't respond to conventional therapies. To avoid overlooking this problem, they recommend that while examining patients with OSD, doctors stay alert for NK risk factors in the patient history and demographics. According to the IRIS Registry, patients with NK are more likely to be women (58.91%), older (mean age at onset: 68), and Caucasian (78.58%).[4] Patients have significantly worse visual acuity at diagnosis if they are older, male, Hispanic or Latino, or of African ancestry.[4]

Clinically, most patients have unilateral NK (58.14%) versus bilateral involvement (16.13%). Visual acuity at diagnosis is worse in patients with unilateral disease or concomitant diabetes, corneal transplantation, or herpetic keratitis.[4] Herpetic keratitis (33.7%) and diabetes (31.6%) are the most common concomitant diagnoses,[4] but there is a host of other potential conditions that influence NK (Figure 1). All of this information can help us shift the focus from treating the manifestations of OSD to instead getting deeper into the underlying problem behind the dryness and blurry vision.

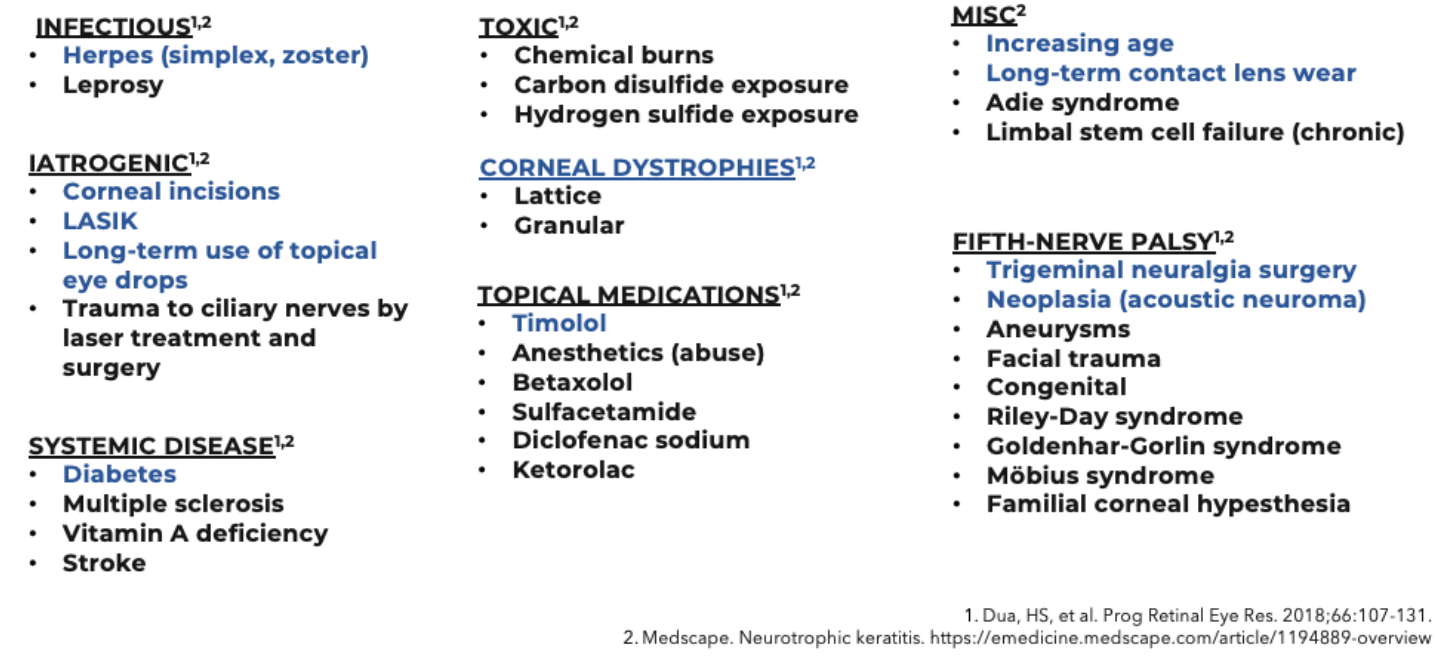


Figure 1. Conditions that may influence NK.

STAGING AND DIAGNOSIS

There was consensus in the NKSG that classification of NK is moving away from the traditional Mackie system.[1] The group arrived at a detailed classification system designed to give clinicians confidence in recommending treatment for earlier stages of NK (Figure 2).

Clinical features in classic Mackie staging are 1) epithelial disease, 2) epithelial defect, and 3) ulceration or perforation. Essentially, a patient can have epithelial disease with some stromal involvement and haziness, after which the epithelium breaks down, and the result is an epithelial defect with stromal opacity that can develop into stromal ulceration and corneal perforation.

To help clinicians identify NK earlier and with greater specificity, the NKSG's staging system ranges from 0 to 6. Zero is altered corneal sensation without evidence of keratopathy. Next, epitheliopathy is divided into two stages, either stage 1 without stromal haze or stage 2 with stromal haze. Stages 3 to 5 are characterized by persistent or recurrent epithelial defects: stage 3 without stromal scarring, stage 4 with stromal scarring, and stage 5 with corneal ulceration. Finally, stage 6 is corneal perforation.

How can we diagnose NK in the two earlier stages of the new classification—stages 0 and 1, which precede Mackie stage 1 epitheliopathy and stromal haze? According to the NKSG, the diagnosis should hinge on corneal sensation testing (in a protocol without anesthetic), ideally across the center and four quadrants, to determine if sensitivity is present, absent, or reduced.[5]

Qualitative testing for corneal sensation: Corneal sensitivity is greatest in the central cornea, which has five to six times as many nerve fibers compared to periphery, dropping rapidly as distance increases from the central cornea and showing greater sensitivity in the temporal limbus than the inferior limbus.[6-8]

Qualitative tests for sensitivity require no new equipment. To see the patient's reaction to stimulation, one can touch the eye in all four quadrants with a wisp at the end of a cotton swab, plain dental floss, or a bit of a tissue (although I find tissue a little too rough). This can be a challenge, in my experience, because patients sometimes indicate that they feel my testing because they can see it in some quadrants, but with experience, I am able to determine if sensation is present, absent, or reduced by watching the patient's response. Do they blink when I tap the eye? How hard do I have to touch the cornea before they respond?

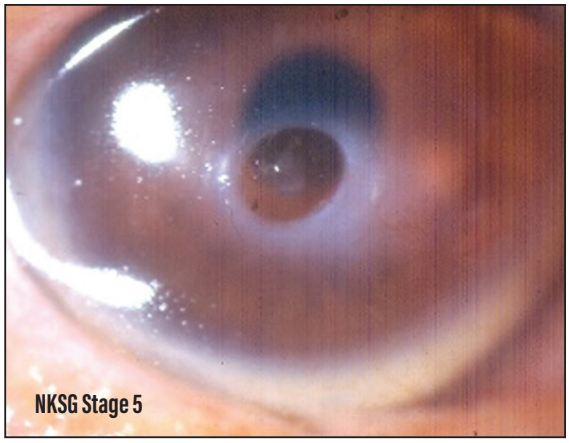
Quantitative testing with an esthesiometer: Esthesiometers that provide standardized, quantitative assessment of corneal sensation are often used in research and clinical trials as well as some practices. With the Cochet-Bonnet esthesiometer, a retractable nylon monofilament is extended to its full length of 6 cm, the filament tip is placed on the eye, and then the filament is retracted in 0.5 cm increments until the patient can feel its contact. The length is recorded, with shorter length indicating decreased sensation, and the process is repeated in the superior, temporal, inferior, and nasal quadrants.[1,11]

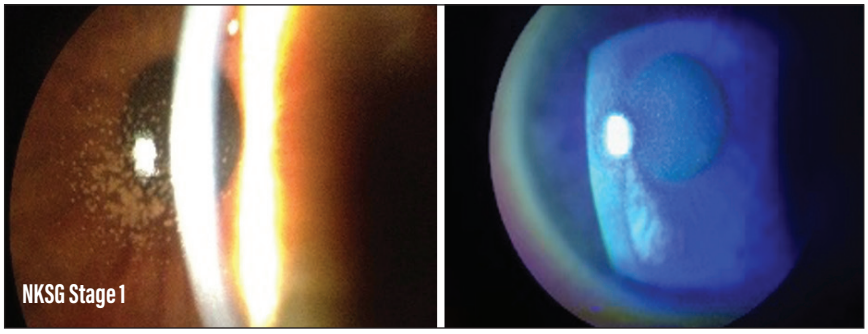
Other esthesiometer options include a portable noncontact device that stimulates the eye using controlled air pulses.[12] It can be handheld or attached to the slit lamp. This newer device (2023) can detect subclinical corneal dysesthesia and related pathologies like diabetic keratopathy or fifth cranial nerve

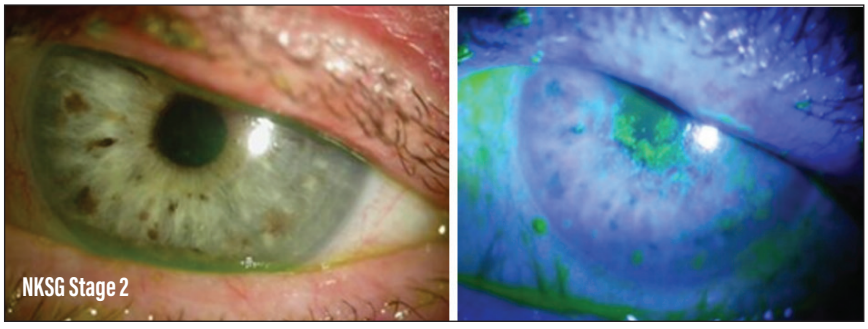
MACKIE	
	Clinical Features
1	Punctate epitheliopathy (punctate corneal fluorescein/ LG staining) Decreased TBUT Stromal haze
2	Persistent epithelial defect with smooth rolled edges Stromal opacity
3	Stromal thinning/ulceration Corneal perforation

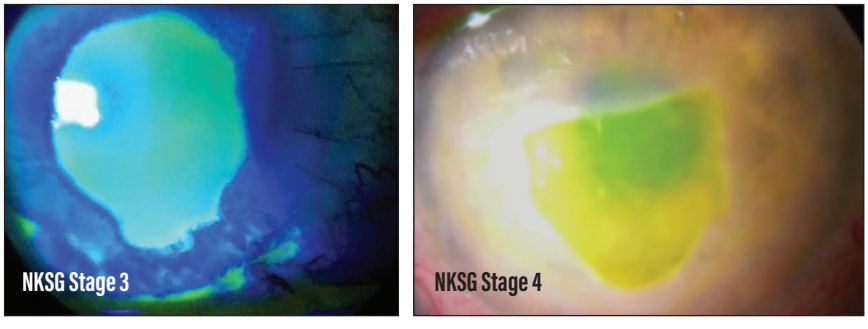
NK STUDY GROUP	
	Clinical Features
0	Altered sensation without keratopathy
1	Epitheliopathy without stromal haze
2	Epitheliopathy with stromal haze
3	Persistent or recurrent epithelial defects
4	Persistent or recurrent epithelial defect and stromal scarring without corneal ulceration
5	Persistent or recurrent epithelial defect with corneal ulceration
6	Corneal perforation

Mastropasqua L, et al. *J Cell Physiol*. 2017;232:717-724.
Neurotrophic Keratopathy Study Group. Neurotrophic keratopathy: An updated understanding. Ocul Surf. 2023;30:129-138









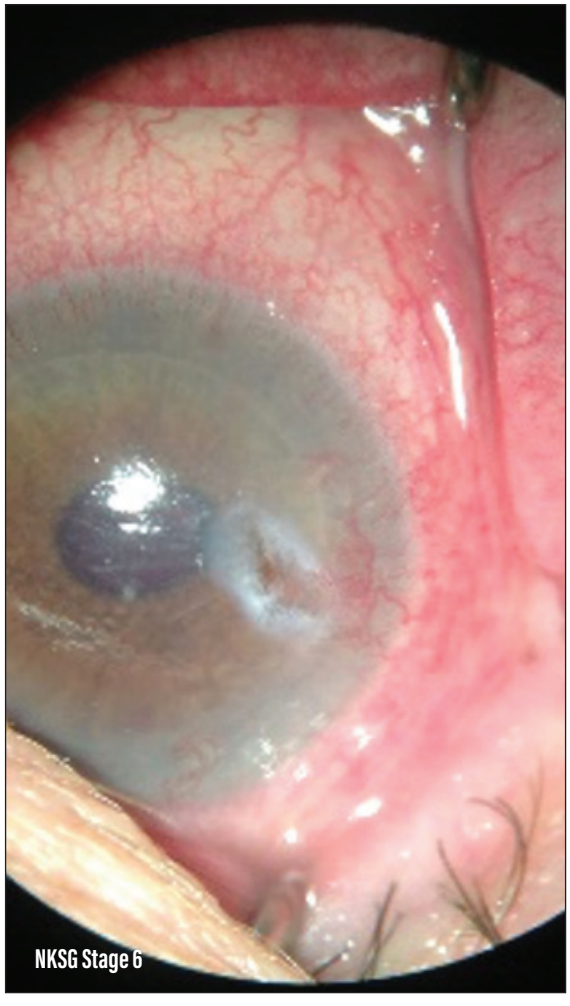


Figure 2. Mackie and NKSG classification systems.

lesions. Another option is a disposable, single-use, esthesiometer that eliminates the need for sterilization after each use. It has proven to have significant agreement ($P = .001$) with Cochet-Bonnet esthesiometer measurements.[13] Metrics for NK diagnosis are clear, with all NK patients showing test values ≤35 mm; NK diagnosis was excluded above 55 mm. The device is simple to operate correctly, making evaluations reproducible (99.6%) between different operators.

Differential diagnosis: Loss of corneal sensation means a patient has NK, but other conditions can influence the nerves,

making differential diagnosis essential. For example, neuropathic pain from corneal neuralgia or keratoneuralgia can cause "pain without stain" in response to minimal or even no stimulus.[3,9] In addition, some diseases with overlapping features of NK (including somewhat reduced corneal sensitivity) can eventually lead to NK: DED, contact lens-related disorders, blepharitis, exposure keratopathy, stem cell deficiency, topical drug toxicity, mild chemical injury, and herpes simplex keratitis.[3,9]

Other diagnostic considerations: Chronic comorbidities can also confound the diagnosis of NK, increasing the need for a thorough diagnostic workup, including a confirmatory test. That workup should include a clinical history, corneal sensitivity testing, complete eye exam (including eyelid and pupillary examination with the slit lamp to rule out diabetic retinopathy, misdirected lashes, and cranial nerve defects), corneal and conjunctival staining with measurement of any epithelial defect, and a Schirmer test to evaluate whether the lack of corneal sensitivity has impaired the quality of blinking and tear production.[9,10] The exam also may include corneal cultures to rule out secondary infection, in vivo confocal microscopy to evaluate affected sub-basal nerves, evaluation for systemic immune disorders, and anterior segment OCT to detect changes in corneal thickness.[9,10]

A CLOSER LOOK AT WHEN TO TEST

According to the NKSG, testing corneal sensitivity is reasonable for patients with 2+ staining, epitheliopathy, and suspected NK symptoms.[5] They also recommend testing at some point during a workup for DED, particularly in patients who have previously tried and failed traditional therapies.

Optimally, when checking for NK, I try to find a correlation between the cause of the decreased sensation and the patient's pathology. I gain a lot of insight from the clinical history—poorly controlled diabetes, previous surgery or trauma, zoster, scarring, or a history of unsuccessful treatment for DED (Figure 3).

There are uncommon cases where the initial exam points to NK, rather than the patient history. For example, for a patient with OSD, I might look at tear osmolarity, a Schirmer test, tear breakup time, and staining on the corneal surface. If I think, "Wow, these eyes should feel terrible," and the patient says, "I don't feel anything," I clearly need to test for decreased sensation. This is true any time the signs are more severe than the patient's level of discomfort would predict.

More often, the first clues about NK come in follow-up visits, when patients aren't responding to treatment. Let's say a patient has a 4-mm epithelial defect because they were poked in the eye. They receive standard treatment for a mild traumatic corneal abrasion, perhaps ointment and a bandage contact lens, but they don't heal in the expected timeframe. They come back in a few days, and they still have a 4-mm epithelial defect. Perhaps they weren't compliant with at-home care, so we give it a few more days. If that defect hasn't healed completely in 1 week, that's a red flag. Even if it's healed to 2 mm, that's not the expected rate of healing. It's likely that healing is compromised by decreased corneal innervation.

SYMPTOMS: HOW PATIENTS EXPERIENCE NK

When we first approach patients, symptomatology is often the first step. What is the patient experiencing? If patients are experiencing blurred vision, we focus on finding the cause. If they're experiencing irritation and discomfort, we focus on making their eyes feel more comfortable. The problem with diagnosing NK based on symptoms is that sometimes patients don't yet

Strongly Recommended	Could be Considered
• Persistent epithelial defect which does not improve within 14 days	• Acquired limbal stem cell deficiency
• Painless, newly observed epithelial defect of unknown etiology	• Newly observed epithelial staining and persistent poorly controlled diabetes
• No pain in the affected eye and multiple, concomitant risk factors, eg, persistent, poorly controlled diabetes, reduced blink, H_x of corneal procedures	• Persistent, poorly controlled diabetes and vision changes due to DR or cataract (even in absence of corneal findings)
• H_x of procedures or conditions that may have affected the trigeminal nerve	
• H_x of herpetic eye disease	

Dana R, et al. BMC Ophthalmol. 2021;21:327.

Figure 3. Corneal sensitivity testing recommendations.

have any classic visual problems or changes in comfort, but they might have underlying progressive disease that will threaten their vision in the future. We see that particularly in patients who have herpes zoster, which might take 2 years to reveal just how severe the damage, pain, and vision problems can get. It's common to think patients have been successful with therapy, and then they are diagnosed with immune keratitis and have to deal with a breakdown of the scar.

How should we approach the relationship between symptoms and NK? What are patients experiencing? In one study, researchers sought to develop a questionnaire to illuminate how patients experience NK and how their experience might help us manage the condition.[14] They acknowledged that NK is a heterogeneous disease that affects patients differently. While clinical trials for eye treatments often include patient-reported outcomes (PROs) to capture symptomatic changes that only the patient can assess, there is no validated NK-specific PRO measure.[14] Their study of 14 patients showed negative impacts on daily activities as well as mental health challenges, including frustration and concern about losing their eyesight.

In one-on-one interviews, patients reported redness (n = 12, 86%), sensitivity to light (n=11, 79%), general discomfort (n=9, 64%), dry eye (n=9, 64%), reduced visual acuity (n=9, 64%), and blurred vision (n=8, 57%). Eye fatigue was also common (n=8, 57%), but only reported by patients after prompting. Symptoms that most impacted daily living were frustration (n=10, 71%), driving impairment (n=8, 57%), reading impairment (n=7, 50%), difficulty watching television (n=7, 50%), and concern with potential vision loss from NK (n=6, 43%). Researchers noted that patients considered other symptoms less important, including redness, burning or stinging, irregular blinking, decreased corneal sensation, tear production, and watery eyes.

This information is helpful in crafting questions about symptoms, including whether patients experience frustration with their eyes and how daily tasks have changed. Rather than discomfort, their symptoms might be variable vision that makes it hard to use their cell phone or stay on the computer for a long time. Their vision fades out as the day goes on and they don't know why.

It's important to ask the right questions to get to these symptoms. For example, many patients go through the history and testing process with technicians in my office, and when I see them in the exam room, I see significant signs of OSD and a history that suggests potential NK involvement. If I ask, "Are you having any problems?" patients answer, "No." But if I ask, "Are your eyes ever dry or red?" or "Do your eyes get blurry sometimes?" they say, "Oh yeah—all the time." Or if I ask, "Does it make you drive less or read less?" they say, "Sure."

It's amazing how often patients tell me, "Yes, my eyes have been red for months." They need to be educated that redness may be a symptom of conditions that can lead to permanent vision loss. Patients have often experienced these symptoms for so long that they think it's normal for them to feel eye fatigue, blurry vision, discomfort, and light sensitivity. This is especially true in older patients, who are accustomed to accepting discomforts as the gradual effects of aging. A thoughtful and proactive approach helps me understand their symptoms.

NKSG UNIVERSAL TREATMENT RECOMMENDATIONS

Historically, NK treatment required a stepwise approach based on Mackie stages of disease severity. Treatment at each stage builds on therapies for the previous stages:[11,15]

- **Mackie stage 1:** External treatments to moisturize and protect the surface are recommended for stage 1 epithelial disease, including artificial tears or ointment, eyelid taping, and punctal plugs. Using cenegermin recombinant human nerve growth factor (rhNGF) is an option.
- **Mackie stage 2:** Additional options for epithelial defect include therapeutic contacts, topical autologous serum, tarsorrhaphy, botulinum toxin injection, topical NSAIDs, and topical steroids.
- **Mackie stage 3:** When an ulcer or perforation has occurred, options added to the list include ulcer culture, amniotic membrane transplant, conjunctival flap, and neurotization.

The NKSG recognized that because NK is a progressive disease, intervention with more aggressive treatments should be considered as an option for patients with significant decreased corneal sensation, even at NK's early stages, to improve the ocular surface permanently or long term and reduce the chances of progression.[1,5] The group specified that patients with NKSG stage 1 NK should receive treatment for both epithelial and neural components, further emphasizing that cenegermin is effective for stage 1 NK and should be considered for patients who do not respond to conventional therapies.[5]

In addition, the NKSG identified universal treatment recommendations regardless of the etiology or severity of NK:[1]

- Clinicians should optimize the ocular surface and remove any factors that can compromise the corneal epithelium.
- Other ocular surface issues, such as blepharitis or meibomian gland disease, lid abnormalities, and chronic infection or inflammation, should be addressed.
- Ocular surface inflammation should be treated with cyclosporine, lifitegrast, or corticosteroids.
- A recent phase 2 clinical trial has assessed the role of cenegermin in patients with DED and demonstrated significant improvements in signs and symptoms after 4 weeks of treatment.[16]

These recommendations not only go farther to prevent disease progression, but they also are a better fit for my actual experiences in practice. It's rare for me to see patients go through the Mackie stages in sequence because they are often already into stage 2 by the time they are symptomatic enough to come see me. Rather than just focusing on the findings of my exam, I'm often trying to uncover etiology that's causing NK to progress, particularly in patients who don't respond to traditional treatments.

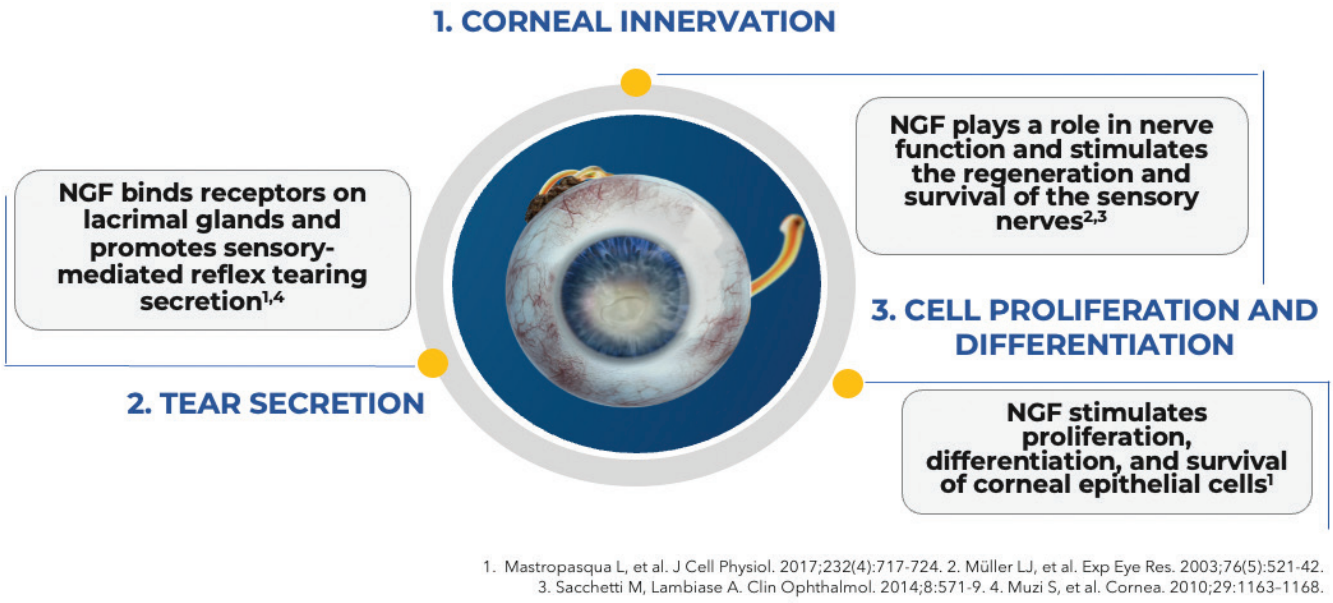


Figure 4. Endogenous NGF maintains corneal integrity by three mechanisms.

For example, I might examine a patient who has chronic keratopathy with a sickly-looking, almost gray epithelium and light, diffuse staining. With more staining in the central cornea, poor response to traditional treatments, and the absence of any obvious cause such as exposure, I know NK may be the problem. Under the NKSG guidelines, this patient can be considered a candidate for an rhNGF. At the same time, as reflected in the NKSG recommendations, I remove all other factors that could influence the ocular surface because someone with NK will not tolerate them well. This often means adding traditional treatments for blepharitis and DED such as punctal plugs, artificial tears, and ointments, and occasionally treating infections, lid malpositions, and other insults.

SERUMS, AMNIOTIC MEMBRANE, AND NEUROTIZATION

As we explore different treatment options used to help patients with significant epitheliopathy and persistent epithelial defects, our primary choices are serums, amniotic membranes, neurotization, and rhNGF. Serums or plasma are an effective choice as a primary or adjunct therapy.[17-22] For patients with significant disease, I often prescribe serum eye drops alone or in combination with scleral lenses. Blood serum can be created in various concentrations based on the severity of a patient's disease, which makes it a good option for my patients who need lower-level maintenance therapy for the ocular surface. They typically get a 6-month supply, usually not covered by insurance.

Studies show the success rates of serum alone (20% to 50% concentration) range from 71% to 100% within 90 days.[17-19] Umbilical cord serum may be more effective and has higher concentrations of substance P and nerve growth factor (NGF) than peripheral blood serum.[20] Plasma rich growth factors healed epithelial defects in 97.4% of Mackie stage 2-3 NK cases after 11 weeks.[21] Serums also can be used safely with silicone hydrogel contact lenses, with no inflammation or deposits.[22]

Amniotic membrane transplant also shows positive success rates in healing neurotrophic ulcers resulting from persistent epithelial defects. In a randomized clinical trial, healing of refractory neurotrophic ulcers was achieved 73% of the time with amniotic membrane transplant versus 67% of the time with conventional therapy (lubrication plus bandage contact lens or tarsorrhaphy).[23] In another study, healing rates for neurotropic ulcers were equivalent with amniotic membrane transplant (73%) and autologous serum (70%).[24] Multiple layers of amniotic membrane are recommended for deep ulcers and descemetoceles.[25]

In corneal neurotization, corneal sensitivity is restored after implanting sural nerve grafts from the leg.[26] The free sural nerve graft is coapted end-to-side with the supratrochlear nerve, and the distal portion of the nerve is separated into fascicles that are distributed around the limbus. Although I offer this option to my patients, none have had the procedure yet because it takes 5 months for sensitivity to return to normal. Nevertheless, it may be particularly advantageous for patients with significant nerve loss, trauma, or surgeries whose fifth nerve may never regenerate.

RECOMBINANT HUMAN NERVE GROWTH FACTOR

NGF is a neurotrophin occurring naturally in the body that stimulates the healing, regeneration, and survival of sensory nerves in the cornea, promotes tear production, and induces corneal epithelial cell proliferation, differentiation, and survival (Figure 4).[9,27,28]

Cenegermin-bkbj, a novel rhNGF that is structurally identical to the NGF protein, was approved by the FDA in 2018 for treatment of all Mackie stages of NK.[29-32] This 20 µg/mL ophthalmic solu-

tion is typically given 6 times a day for 8 weeks. Previous clinical trials showed cenegermin was safe and effective for patients with Mackie stage 2 and 3 NK.[33,34]

As we shift toward following the NKSG recommendations for early, aggressive treatment of NK, another clinical trial published this year showed that cenegermin is just as effective for patients with less advanced disease in increasing corneal sensation and improving visual acuity.[35] In the clinical trial, 37 patients with Mackie stage 1 NK were given one drop of cenegermin 20 µg/mL, 6 times a day for 8 weeks and monitored at 4, 8, and 32 weeks.[35] Corneal epithelial healing was observed in 84.8% of patients (95% CI 68.1-94.9%; $P < .001$) at week 8. Corneal sensitivity showed improvement in 91.2% of patients at week 8 and 82.1% of patients through week 32. At week 8, patients gained a mean change in best-corrected distance visual acuity of 15 letters.

At least one adverse event was reported by 73.0% of patients at 8 weeks and 45.7% at week 32.[35] The most common adverse event was eye pain (37.5%), followed by blurred vision (10.8%) and eyelid pain (8.1%). Most adverse events were mild or moderate and were only reported during the treatment period. No serious adverse reactions related to treatment occurred in any trial of cenegermin.[34,36,37] Most were mild and transient ocular reactions that did not require treatment discontinuation or any corrective treatment. About 16% of patients experienced eye pain following instillation, and 1% to 10% experienced corneal deposits, foreign body sensation, ocular hyperemia, ocular inflammation, and tearing.[34,36,37]

Before starting treatment with cenegermin, I counsel patients that there's about an 85% chance of success in regenerating nerves and improving corneal sensation, but that means they will begin to feel pain and irritation from cell damage that has occurred to the ocular surface. Some patients don't like feeling irritation for the first time, and even normal sensations like eye drops can feel strange. I explain that therapy will help the ocular surface to heal and, ultimately, feel better. The hope is that early treatment can help prevent NK from progressing.

The NKSG recommends maintenance therapy that delivers ongoing nutrition to corneal nerves, but it points out that this remains an unmet need.[5] New therapies are being investigated for this purpose, some of which should soon have published results, including varenicline nasal spray, human recombinant dHGF, udonitrectag synthetic peptidomimetic of NGF, Thymosin β4 0.1%, and topical insulin.[38]

NK CASE STUDIES

To illustrate specific ways we can apply what we know about NK in real situations, I'd like to share two cases. A short time ago, I examined a 59-year-old African-American woman who had a previous herpes zoster OD. The patient had uveitic glaucoma (IOP 12 mm Hg), keratoconjunctivitis sicca, corneal scarring, previous cataract surgery, and NK. Her VA was 20/40 OD. She'd begun developing epitheliopathy with a 5% paracentral epithelial defect, subepithelial corneal scarring, and diffuse 1+ edema.

This patient was treated with punctal plugs, lubrication, and a bandage contact lens, but what ultimately healed her eye was treatment with cenegermin. It's important to remember that when you examine a patient who has herpes zoster, previous surgery, uncontrolled diabetes, or other known risk factors for NK, the lack of innervation is likely to play a role in problems on the ocular surface. After successful treatment, many zoster patients return at progressive follow-up intervals, allowing us to catch problems early. We need to examine the ocular surface and check their corneal sensation, even if they're asymptomatic, so we can diagnose and treat NK early. Some patients, desensitized by NK, wait to see a doctor until their eye reaches an acute phase. In these cases, I determine how much of the cornea is involved and how much corneal sensation is affected, as well as if uveitis is causing a delayed inflammatory reaction.

In another interesting case, a 75-year-old white man with recurrent herpes simplex OS presented complaining of foreign body sensation, tearing, and redness for 5 days. In addition to the virus, his history included coronary artery disease, NK and corneal scarring OS, primary open-angle glaucoma, keratoconjunctivitis sicca, and posterior blepharitis. He'd had punctal plugs as well as an Ahmed valve and selective laser trabeculoplasty OS. His IOP was 16/23 mm Hg, and VA was hand movements OS and 20/30 OD (down from his normal 20/20 OD). Slit lamp examination showed the following features:

- OS: 2+ meibomian gland dysfunction, lid margin thickness, punctal stenosis on upper lid, punctal plug in the lower lid, faint corneal edema, subepiretinal scarring, 2+ peripheral neovascularization, posterior chamber intraocular lens
- OD: 1+ meibomian gland dysfunction, lid margin thickness, punctal stenosis, 2+ conjunctival injection, dendrite with epithelial defect, posterior chamber intraocular lens

This patient had developed significant NK (Mackie stage 3, NKSG stages 5 and 6), and he came in very late in the disease process. One eye was effectively lost to glaucoma, significant corneal scarring, and other factors. At this point, he had developed a dendrite on his fellow eye after taking a holiday from his maintenance antivirals. This patient was at serious risk for blindness. How could I save his fellow eye, whose VA had decreased from 20/20 to 20/30?

This monocular patient with early-stage NK in the fellow eye needed aggressive, simultaneous treatments for his most pressing, sight-threatening conditions. I ordered systemic antiviral treatment for his herpetic disease. To attempt to alter the course of NK by restoring nerve function, I prescribed cenegermin rhNGF. Following the NKSG recommendations to address the patient's OSD, I also prescribed cyclosporine drops, Omega-3 supplements, and preservative-free artificial tears.

SETTING THE STAGE FOR THE FUTURE

Thanks to the work of our peers in the NKSG, we've gained valuable insights into NK. With a new definition and detailed stages linked to current therapies, we're positioned to better understand

the role of NK. By making corneal sensitivity testing part of an OSD workup, particularly for patients who have not responded to traditional therapies, I think we will find more patients with NK early in the disease process. When we combine that paradigm shift with an rhNGF, it becomes possible to not only treat the effects of NK, but also restore some of the lost innervation for long-term success. The future is looking brighter for patients with NK. ■

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Understanding the Signs, Symptoms, and Latest Therapeutic Recommendations for NK

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Expiration Date: December 6, 2025

INSTRUCTIONS FOR CREDIT

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DEMOGRAPHIC INFORMATION

Profession
___ MD/DO
___ OD
___ NP
___ Nurse/APN
___ PA
___ Other

Years in Practice
___ >20
___ 11-20
___ 6-10
___ 1-5
___ <1

Patients Seen Per Week (with the disease targeted in this educational activity)
___ 0
___ 1-15
___ 16-30
___ 31-50
____ >50

Region
___ Midwest
___ Northeast
___ Northwest
___ Southeast
___ Southwest

LEARNING OBJECTIVES

Did the program meet the following educational objectives?	Agree	Neutral	Disagree
Describe the etiologies, comorbid diseases, surgical issues, and patient-specific factors that may heighten suspicion for neurotrophic keratitis (NK)	_____	_____	_____
Analyze the relationship between different corneal components that underscores the pathophysiology of NK	_____	_____	_____
Perform diagnostic evaluations that distinguish between dry eye and NK	_____	_____	_____
Employ classification/staging systems for NK in facilitating early diagnosis and intervention	_____	_____	_____
Identify the clinical goals of managing NK by assessing the holistic impact on patients, including disease burden and patient experience	_____	_____	_____

POSTTEST QUESTIONS

Please complete at the conclusion of the program.

1. Based on this activity, please rate your confidence in your ability to employ classification/staging systems for neurotrophic keratitis (NK) in facilitating early diagnosis and intervention (based on a scale of 1 to 5, with 1 being not at all confident and 5 being extremely confident).

a. 1
b. 2
c. 3
d. 4
e. 5

2. Which of the following factors is NOT an etiology of NK?

a. Long-term contact lens wear
b. Short-term use of topical eye drops
c. Increasing age
d. Diabetes

3. A 64-year-old patient presents with blurred vision in his right eye for 2 months. He has a history of hypertension, congestive heart failure, and facial trauma. Punctate epitheliopathy without stromal haze is observed during the slit-lamp examination. Which patient-specific factors increase the suspicion for NK?

a. Facial trauma and epitheliopathy
b. Hypertension and facial trauma
c. Hypertension, facial trauma, and epitheliopathy
d. Congestive heart failure and facial trauma

4. NK is characterized by the breakdown of the corneal _____ and impaired corneal innervation of the _____ nerve.

a. Epithelium, facial
b. Epithelium, trigeminal
c. Stroma, facial
d. Stroma, trigeminal

5. Which of the following diagnostic tests is used to distinguish between dry eye disease and NK?

a. Corneal sensitivity
b. Corneal staining
c. Schirmer test
d. Tear film osmolarity

6. A 58-year-old patient presents with symptoms of dryness. She has a history of poorly controlled diabetes and has used artificial tears for years. She is a nonresponder to lifitegrast and cyclosporine ophthalmic solutions. Which of the following diagnostic strategies is most appropriate?

a. Perform a workup for dry eye that includes corneal staining and Schirmer testing without use of topical anesthetic
b. Perform a workup for dry eye that includes corneal staining and Schirmer testing with use of topical anesthetic
c. Perform a workup for dry eye that includes corneal staining and sensitivity testing without use of topical anesthetic
d. Perform a workup for dry eye that includes corneal staining and sensitivity testing with use of topical anesthetic

7. NK observed as corneal perforation is graded as stage _____ and stage_____ according to the classification of Mackie and the Neurotrophic Keratitis Study Group (NKSG), respectively.

a. 1, 3
b. 2, 3
c. 2, 4
d. 3, 6

8. A 72-year-old patient presents with blurred vision. During the slit-lamp examination, a central epithelial defect with stromal scarring and corneal hypoesthesia are observed in his left eye. According to the NKSG classification, what is the stage of NK?

a. 3
b. 4
c. 5
d. 6

9. A 54-year-old patient presents with blurred vision and redness. During the slit-lamp examination, punctate epitheliopathy with stromal haze and corneal hypoesthesia (Mackie Stage 2) are observed in her left eye, and punctate epitheliopathy without stromal haze and corneal hypoesthesia (Mackie Stage 1) are observed in her right eye. Telangiectasia and anterior blepharitis are observed in both eyes. According to the NKSG, which of the following treatment recommendations is most appropriate?

a. Recommend a pressure patch and topical antibiotic for left eye
b. Recommend a silicone hydrogel bandage contact lens and topical antibiotic for left eye
c. Recommend a silicone hydrogel bandage contact lens, topical antibiotic, and topical corticosteroid for left eye
d. Recommend a silicone hydrogel bandage contact lens for left eye and a topical antibiotic and topical corticosteroid for both eyes

10. A 2020 study by Murray et al examined the patient experience of NK using a questionnaire and found that patients with NK reported the ocular symptom of _____ only when queried.

a. Eye fatigue
b. Sensitivity to light
c. General discomfort
d. Blurred vision

11. A 78-year-old patient presents with symptoms of dry eye, photophobia, and a loss of interest in driving. In the past, he has been nonresponsive to topical prescription medications for dry eye and preservative-free artificial tears. During the slit-lamp examination, meibomian gland dysfunction, punctate epitheliopathy without stromal haze, and corneal hypoesthesia (Mackie Stage 1) are observed in both eyes. Which of the following management approaches is most appropriate?

a. Recommend thermal pulsation light therapy, blepharoexfoliation, and follow-up in 6 weeks
b. Recommend thermal pulsation light therapy, blepharoexfoliation, an artificial tear ointment at night, and follow-up in 6 weeks
c. Recommend thermal pulsation light therapy, blepharoexfoliation, punctal plugs, and follow-up in 6 weeks
d. Recommend thermal pulsation light therapy, blepharoexfoliation, topical cenegermin ophthalmic solution, and follow-up in 6 weeks

ACTIVITY EVALUATION

Your responses to the questions below will help us evaluate this activity. They will provide us with evidence that improvements were made in patient care as a result of this activity.

Rate your knowledge/skill level prior to participating in this course: 5 = High, 1 = Low_____

Rate your knowledge/skill level after participating in this course: 5 = High, 1 = Low_____

This activity improved my competence in managing patients with this disease/condition/symptom. _____ Yes _____No

Probability of changing practice behavior based on this activity: _____High _____ Low _____No change needed

If you plan to change your practice behavior, what type of changes do you plan to implement? (check all that apply)

Change in pharmaceutical therapy _____
Change in nonpharmaceutical therapy _____
Change in diagnostic testing _____
Choice of treatment/management approach _____
Change in current practice for referral _____
Change in differential diagnosis _____
My practice has been reinforced _____
I do not plan to implement any new changes in practice _____

Please identify any barriers to change (check all that apply):

_____ Cost
_____ Lack of consensus or professional guidelines
_____ Lack of administrative support
_____ Lack of experience
_____ Lack of time to assess/counsel patients
_____ Lack of opportunity (patients)
_____ Reimbursement/insurance issues
_____ Lack of resources (equipment)
_____ Patient compliance issues
_____ No barriers
_____ Other. Please specify: _____

The design of the program was effective for the content conveyed	___ Yes	___ No
The content supported the identified learning objectives	___ Yes	___ No
The content was free of commercial bias	___ Yes	___ No
The content was relative to your practice	___ Yes	___ No
The faculty was effective	___ Yes	___ No
You were satisfied overall with the activity	___ Yes	___ No
You would recommend this program to your colleagues	___ Yes	___ No

Please check the Core Competencies (as defined by the Accreditation Council for Graduate Medical Education) that were enhanced through your participation in this activity:

_____ Patient Care
_____ Practice-Based Learning and Improvement
_____ Professionalism
_____ Medical Knowledge
_____ Interpersonal and Communication Skills
_____ System-Based Practice

Additional comments:

_____

_____

This information will help evaluate this activity; may we contact you by email in 3 months to inquire if you have made changes to your practice based on this activity? If so, please provide your email address below.

_____

_____

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